ORIGINAL ARTICLES

CHANGING CONCEPTION OF CORONARY ARTERY DISEASE*

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WITHIN the memory of most of us the concepts of the pathogenesis, the functional and structural pathology, and the clinical characteristics of coronary artery disease, have undergone remarkable changes. The disease was first recognized in the latter part of the eighteenth century. Heberden gave the original description of angina pectoris in 1786. Harvey described myocardial infarction with cardiac rupture in his report on the heart of George II. Fothergill in 1776, and Black, Jenner and Parry (John Hunter's heart), as well as Baillie at the beginning of the nineteenth century, discussed coronary arteriosclerosis in relation to angina pectoris. In 1809, Burns of Glasgow compared the pain in this condition to that experienced in an extremity below a ligated artery. Earlier descriptions of the lesions had been given by Drelincourt, Thebesius, and Bellini. Kreysig in Germany and Testa in Italy made further contributions to the subject in the middle of the nineteenth century, but Laennec and many clinicians after him slowed progress by asserting that angina pectoris was a nervous disease. In 1842 Marshall Hall associated coronary artery disease with sudden death, and Ericson was the first to produce sudden death experimentally by ligating the coronary arteries of dogs. Virchow demonstrated the phenomenon of coronary embolism, and Weigert, in 1882, described the infarction followed by blocked coronary arteries in man. René Marie in 1896, Sternberg[1] in 1914, and others in the interval, gave excellent pathological descriptions of coronary infarction.

THEORIES CONCERNING PATHOLOGY

Cohnheim and von Schultness-Rechberg,[2] in 1881, made the assertion that, functionally, the coronary arteries were end-arteries. This viewpoint was supported by Wiggers,[3] Porter,[4] Saphir, Priest, Hamburger, Katz[5] and others, and more recently by Blumgart, Schlesinger and Davis.[6] In conflict with this conception was the early demonstration of coronary interarterial communication by Ruysch in 1704 and Lower in 1728, which was confirmed by many workers, including such recent workers as Gross,[7] Hirsch and Spalteholz,[8] and Spalteholz and Campbell,[9] who believed that these communications were functionally adequate. Pratt,[10] and later Wearn and his associates,[11,12] and others demonstrated the possible functional communications of the "Thebesian" system[12] with the coronary arteries and capillaries, and as early as 1880 Langer[13] had demonstrated the collateral supply to the coronary system from the extracardiac vessels of the pericardium, the bronchi, the diaphragm, and the great vessels. The history of the clinical appreciation of acute coronary artery occlusion began with the work of Hammer[14] of St. Louis in 1876, who first made the bedside diagnosis. However, it remained for Dock[15] in 1896, Abrotzow and Strachsika[16] on 1910, and Herrick[17] in 1912 to define the criteria for diagnosis of this condition, or, more precisely, of acute myocardial infarction.

Although most of the important pathological and experimental conditions found in coronary artery disease had been described, the practicing physician at the beginning of the twentieth century still was confused by the varied opinions on the causes of angina pectoris and of so-called chronic myocarditis with heart failure. The pain of angina pectoris may in rare instances not be due to coronary artery disease; however, this condition is now generally accepted to be its cause. Sir Clifford Albutt and others, by supporting the "aortic" theory of angina pectoris, slowed the popular acceptance of such ideas. Although more complete details of the clinical picture of acute coronary artery occlusion appeared in the literature during the first two decades of this century (for instance, Libman and Sacks[18] reported leukocytosis and Fred M. Smith[19] the alterations in the electrocardiogram), the diagnostic criteria were not generally known and the frequency of the occurrence of the disease was not recognized. Until the beginning of the third decade the diagnosis was rarely made, and the criteria were not even taught in the medical schools of America and Europe. What gave the tremendous impetus to the recognition of this lesion as the commonest nonaccidental cause of sudden death in man is not known. Perhaps the repeated articles of Herrick and of Levine were responsible. The diagnosis of acute indigestion commonly made during and prior to the early 1920's is practically unknown today, since in most of the cases formerly so diagnosed we now find acute coronary artery occlusion with infarction.

CHART I

Chart 1 outlines the findings in a case of acute coronary artery occlusion eleven years after Herrick's[17] description, with which this case is compared. It illustrates that even a typical case observed by several prominent physicians as late as 1922 was diagnosed erroneously. From 1925 to 1930 the knowledge of the diagnostic criteria rapidly infiltrated this country, and by the beginning of the fourth decade the characteristic picture of acute coronary artery occlusion was well recognized. Likewise, the conception that angina pectoris is a disease almost exclusively due to anoxia of the myocardium, as restated in the article of Keefer and Resnik,[20] was accepted by most physicians.

The first demonstration of the value of the electrocardiogram in the diagnosis of acute coronary artery occlusion was made by Fred M. Smith[19] in 1918, when he compared the records of dogs with ligated coronary arteries with those of patients who had coronary artery occlusion. Pardee[21] in 1920 demonstrated the significance of the S-T interval and the T-wave changes; Wolferth and Wood[22] in 1932 indicated the diagnostic im-

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CHART 1.—*Comparison of Case of Mr. X With Herrick's Case 1*

Case 1—1912—aet. 55	Mr. X—1923—aet. 58*
1. One hour after meals—pain in lower precordial region. Fairly abrupt onset. Occurred at rest.	1. Distress. ?Lower chest and epigastrium during night after dinner. Fairly abrupt onset. Occurred at rest.
2. Nauseated and induced vomiting. Later projectile vomiting.	2. Nauseated. Vomiting.
3. Pain persisted 3+ hours.	3. Pain through night and recurred through second and third days. Cleared fourth day.
4. Exhaustion —. Weak pulse.	4. Exhaustion during second day. Circulatory collapse third, fourth and fifth days.
5. Cold with sweating +.	5. Cold with sweating +.
6. Moist râles right and left chest. ?cough. ?rapid respiration.	6. Moist râles right and left chest. Cough +. Respiration 40. Cheyne Stokes respiration.
7. Fever second day.	7. Fever, second and sixth days to 102°. Cleared by sixth day.
8. Moderate cyanosis.	8. ? cyanosis.
9. Tachycardia 140.	9. Tachycardia 120-130.
10. Despite weakness able to undertake physical activity. Mind clear.	10. Very active first day and never at complete rest for two and one-half days. Mind clear.
11. Sudden death in fifty-two hours.	11. Sudden death on sixth day.
12.	12. Previous history of hypertension and angina pectoris.
13.	13. Sister died suddenly of similar disease. ? other family members.
14.	14. Leukocytosis.
DIAGNOSIS: (?) Hypertension. Coronary arteriosclerosis. Acute coronary artery occlusion with infarction. Sudden death.	Hypertension. Angina pectoris. Acute G. I. attack. Crabmeat-copper ptomaine poisoning. ? Gall-bladder disease. Pneumonia. Circulatory collapse. Apoplexy.

* From official medical bulletins.

portance of Lead IV; and Wilson, McLeod, Barker and Johnston[23] were the first to report the localization of certain lesions by the characteristic "Q1-T1" and "Q3-T3" forms of the electrocardiogram. However, excellent reviews by Wilson[24] and others have shown that many causes other than acute coronary artery occlusion may produce approximately the same electrocardiographic abnormalities. Therefore, in spite of the recognized importance of electrocardiographic criteria, characteristic changes in the records cannot be accepted as pathognomonic of this condition.

By the middle of the fourth decade the diagnosis of "acute indigestion" had almost disappeared, and coronary artery occlusion was recognized not only by the physician, but also by the lay public. A trend toward overdiagnosis of coronary artery occlusion rapidly followed. Among the conditions falsely diagnosed as such were: acute abdominal disease, dissecting aneurysm of the aorta, acute pulmonary artery occlusion, pleurisy, nerve root pain, spontaneous pneumothorax, esophageal and mediastinal diseases, and the onset of diabetic coma.

PHYSIOLOGIC RELATIONSHIPS

The excellent experimental work of Anrep,[25] Wiggers and Cotton,[26] Katz, Weinstein and Jochim[27] and others on the physiology of the coronary circulation demonstrated the importance of aortic pressure in maintaining adequate blood flow and the constricting influence exercised by the contracting cardiac muscle on the coronary vessels. The earlier end-artery hypothesis had been discarded. Attention was directed to the existence of collateral circulation by the injection studies of Gross,[7] Spalteholz and Campbell[9] and others, and this in turn led to the efforts of Beck[28] and O'Shaughnessy,[29] in the late 1930's, to establish artificial collateral circulation through extracardiac tissue grafts. Prior to this time (and even up to the present) surgical procedures for the relief of angina pectoris were directed toward interrupting the nervous pathways, chiefly those of the afferent cardiac nerves. The assumption was that by this method the unbalance between the demand of the cardiac muscle for oxygen and its available supply might be corrected. Blumgart, Levine, and Berlin[30] introduced the practice of thyroidectomy as a therapeutic measure. When symptoms of anginal or congestive failure could not be relieved by placing the body at relative rest, they attempted to reduce the work of the body still further by lowering the metabolic demands on the heart for the delivery of blood. Only moderate success was achieved.

PROGNOSIS

The extremely grave prognosis attributed to coronary artery occlusion at the time of its first widespread recognition has been modified gradually until, at the close of the fourth decade of this century, only 15 to 25 per cent fatality was attributed to the first attack of myocardial infarction. However, there did seem to be an alarming rise in frequency of the condition as well as of all coronary artery disease. Cohn[31] and others showed that this was probably due largely to (1) the survival of persons into the age of senescence who formerly succumbed to infections such as typhoid fever (which are now relatively infrequent) and (2) the changing habits of diagnosis and diagnostic terminology of the general medical practitioners.

OTHER STUDIES

The apparent increase in coronary arteriosclerosis awakened medical and public consciousness especially to the frequency of cases occurring before the age of fifty years, and resulted in attempts to determine the pathogenesis of such arteriosclerosis. The earlier experiments of Ignatowsky[32] in 1908, in which he produced arteriosclerosis in herbivora by feeding cholesterol, have been supplemented recently. Likewise, by comparing the dietary habits of these animals with the relative dietary habits of different groups of men, Leary,[33] Joslin,

Root, White and Marble,[34] and others concluded that ingestion of cholesterol in eggs, cream and similar foods may play an important part in causing atherosclerosis in man. These conclusions were strengthened by reports such as those of Thomas[35] and Aschoff[36] on the effect of low-fat diets during the World War, 1914-1918, in reducing the incidence of arteriosclerosis in Germany, and of Oppenheim[37] on the occurrence of marked arteriosclerosis in only 11 per cent of the Chinese in Shanghai, whose diet is low in dairy products. Another means of studying this condition is provided by the fact that ordinary clinical atherosclerosis resembles xanthomatosis associated with cholesterolemia. Muller[38] reported this condition in seventy-six persons, and indicated that its occurrence is much more common than was previously believed. In spite of such positive evidence, the relation of foods to arteriosclerosis in man is not generally considered as conclusively proved.

Hypertension is a known cause of arteriosclerosis, but its cause in turn is still unknown, although much work on constriction of the renal artery has been done by Goldblatt[39] and others, which indicates that restricted blood flow through the kidneys produces a hypertensive substance. Diabetes is, likewise, a common cause of premature arteriosclerosis.[34]

Surveys of the racial characteristics and habits of man, made by Glendy, Levine and White,[40] indicate that the pace of modern life may produce premature arterial degeneration, especially in the emotionally high-pitched races. This tendency is known to be inherited chiefly through the mother, and coronary and generalized arteriosclerosis may be on the increase in young persons. Furthermore, tobacco is suspected of causing premature arteriosclerosis in man, although no confirmation of this belief has been universally accepted.

Diseases other than typical arteriosclerosis, recognized as being rare causes of coronary artery narrowing, are: Buerger's disease, xanthomatosis (previously mentioned), periarteritis nodosa, rheumatic or syphilitic arteritis, gummata, neoplasms and unspecified types of thrombotic diathesis.

PRESENT STATUS

As we enter the fifth decade of the twentieth century we find that some of the work of the past fifteen years fits into the mosaic of current opinion. The experimental work on dogs by Beck[28] in 1932, and Burchell[41] in 1940, demonstrated that artificial collateral circulation developed only when the coronary arteries were constricted, and thus confirmed the assumption of Hudson, Moritz, and Wearn[42] that a similar situation existed in the diseased coronary vascular system.

From such work as that of Kountz and Smith[43] on perfused revived human hearts, we conclude that if the blood supply is rapidly diminished to an inadequate flow, infarction may be produced without occlusion of a coronary artery. Gross and Sternberg[44] and others have shown that infarction may occur as a result of increased demand for blood by the cardiac muscle even without extensive narrowing of the coronary arterial aperture. The probable improvement in oxygen supply to the myocardium by favorably changing the circulatory mechanisms in certain persons with postural defects and emphysema was accomplished by Kerr, Cannon, and Lagen[50] through the use of abdominal belts. They demonstrated not only clinical improvement in patients with angina pectoris, but likewise improvement in certain tests of circulatory efficiency, such as measurement of the circulation rate. Careful pathological and clinical correlations culminating in the recent studies of Blumgart, Schlesinger, and Davis[6] have confirmed that this rapid development of unbalance between demand and supply of blood, which produces infarcts, and patchy myocardial necrosis, is caused by a slowly developing inadequacy of the blood supply of the myocardium. They have also demonstrated the remarkable development of collateral circulation in good-sized vessels (over 40 in diameter) on the gradual narrowing of the regular channels, so that often complete occlusions are observed at autopsy in hearts in which no clinical picture of acute infarction had been noted. Usually two or more occluded vessels result in symptomatic anginal or congestive failure.

CHART 2

Chart 2 is given to summarize briefly the elements tending to affect unfavorably the balance of myocardial demand for oxygen and the available supply. Some of these factors have been discussed previously in this paper. If somewhat broad conclusions are drawn from these data, the occurrence of a "silent" coronary artery occlusion would be suspected on the sudden development of angina pectoris or congestive heart failure without other cause. This condition is estimated by certain workers to occur one and one-half to two times as frequently as the occlusions producing the characteristic clinical picture of myocardial infarction. The suggestion follows that, in order to prevent the increase of oxygen demand by the cardiac muscle, patients should be kept quiet when such occlusion is suspected until adequate collateral circulation has developed. The demonstration that collateral circulation develops slowly with demand likewise explains occasional slow spontaneous "cures" in cases of angina pectoris. Thus we must now recognize not only that coronary occlusion induces myocardial infarction, and that myocardial infarction may occur without coronary occlusion, but also that either or both of these conditions, when they occur slowly, may produce the characteristic clinical picture of angina pectoris or congestive heart failure in contrast to the clinical findings which appear with the abrupt development of a myocardial infarct.

Winternitz, Thomas, and LeCompte[45] have described subintimal hemorrhages in the coronary arteries which precede occlusion, having been caused by rupture of one of the extensive groups of vessels adjacent to the atheromatous deposits in the arteries. These hemorrhages may explain the occurrence in about 50 per cent of all cases of acute coronary artery occlusion, of a warning premonitory pain which is felt for several hours

CHART 2.—*Concept of Coronary Circulatory Efficiency Balance Between Myocardial Demand for Oxygen and Available Oxygenated Blood.*

Factors Disturbing Balance:

I. Fall in coronary artery pressure.
   1. Coronary artery disease or congenital anomalies.
   2. Fall in aortic mean blood pressure—aortic insufficiency and stenosis, and congestive heart failure and shock (i. e., surgical). Fall in blood volume or mechanical defects in return of blood to heart.
   3. Tachycardia.
   4. Hypertension—increased perivascular muscle pressure—inadequate capillary supply.
   5. Reflexes causing coronary artery spasm—abdominal disturbances, gall-bladder disease, psychic factors, cold, etc.

II. Increased demand without adequate increased supply of blood.
   1. Hypertrophy of myocardium.
   2. Valve lesions and hypertension increasing work.
   3. Hyperthyroidism or thyroid administration.
   4. Epinephrin—through more forceful contraction and increased peripheral arterial pressure.

III. Oxygen content of the blood.
   1. Anemia.
   2. Anoxic anoxemia, i. e., high altitude, poor pulmonary aeration of blood, veno-arterial shunts, and carbon monoxide poisoning.

IV. Drugs and toxins affecting coronary artery lumen, i. e., pitressin.

V. Inadequate collateral circulation.

A trend toward adequate collateral circulation develops when pressure in a collateral vessel is higher than in the distal portion of the occluded or narrowed artery. Infarction develops if such circulation is not available when a major inadequacy of blood suddenly occurs. Congestive heart failure and angina pectoris develop when the collateral circulation is relatively inadequate, and with a slow development of this blood supply such evidences of failure are often accompanied by the occlusion of one or more coronary artery branches, even though the signs and symptoms of acute infarction have not developed.

to several days before the occlusion takes place (Feil [46] and Sampson and Eliaser [47]).

WORK FOR THE FUTURE

As we look into the future, we are confronted with certain problems that are worthy of statement and conjecture. We must look broadly at every case of angina pectoris or congestive heart failure caused by coronary arteriosclerosis. We must consider the transient or permanent factors which cause relative oxygen insufficiency of the cardiac muscle. We must attempt by the examination of clinical histories and laboratory and electrocardiographic criteria to determine what factor or factors have brought about the condition. With greater clinical acuity we should be able to detect most cases of "silent" coronary occlusion, as well as those of slowly or rapidly developing infarction which do not present characteristic clinical patterns. The future treatment of such cases will be advantageously modified only by our increasing ability to diagnose these pathological changes. We hope that a new study of criteria will be made which will enable us to determine just how completely the collateral circulation has developed and how extensively the natural circulation has been impaired.

According to the recent work of Mallory and his associates,[48] structural recovery of small infarcts takes place in two weeks, that of large infarcts in five weeks; no cardiac rupture ever occurs after the second week. Perhaps many of our patients with characteristic infarction are being confined too long. On the other hand, many patients may require the prolonged limitation of activity which we have given only to those with the characteristic syndrome of acute coronary infarction as clinically recognized. It has been shown that mortality is higher, and dilatation and thinning of the infarcted areas occurs more readily in dogs with experimental infarction that are allowed free activity than in those that are confined for six days after the occlusion.[49] This is added evidence in support of the rest treatment of myocardial infarction.

CALIFORNIA HEART ASSOCIATION AND OTHER AGENCIES

Not only private investigators, but also the United States Public Health Service and various health departments throughout the country are taking cognizance of both rheumatic heart disease and coronary heart disease as major public health problems, and are making efforts to eliminate them. Certain special activities of the American Heart Association may prove valuable in this public health crusade. This organization has recently attempted through a questionnaire to assemble and to analyze critically the widespread individual experiences of over two hundred physicians in regard to the course, treatment, and results obtained in cases of coronary artery sclerosis. These data should aid in evaluating the present methods of caring for patients with this disease. The California Heart Association is undertaking surveys on the incidence of heart disease, and also is making efforts to analyze the influence of industrial strains on hastening its advance. It is also sponsoring studies of the socio-economic problems arising in the adjustment of such cardiac patients to employment.

That so little has been accomplished to date in decreasing the incidence of arteriosclerosis among the general populace is no reason why we should consider the outlook as hopeless. It may be safely predicted that the concerted efforts of the individuals and agencies working in this field will bring about a decrease in the incidence of coronary artery disease, especially in persons of the fourth and fifth decades of life, and will result in the prolongation of the life span of those now suffering from it.

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FIBROMA OF THE NASOPHARYNX*

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ALTHOUGH fibroma of the nasopharynx is of rare occurrence—about one in 164,000 ear, nose, and throat cases—we need generally disseminated information concerning it; otherwise, it is a real emergency when we meet it, and, as treatment has evolved in recent years, we think it worth while to write of it at the present time.

Nasopharyngeal fibroma is usually spoken of as histologically benign, but clinically malignant. Ferreri, quoted by Clark,[1] says that "true pharyngeal fibroma has its origin in the aponeurosis of the spheno-occipital recesses, and sometimes attains enormous size, and by anatomical interference, may produce pressure disturbance, sometimes quite serious, occlude the upper respiratory passages, or penetrate the skull and produce symptoms of hypophysial tumor." It may penetrate the skull through the cribriform plate of the ethmoid bone, or through the pharyngeal vault by way of the sphenoid sinus.

They are attached very firmly to the occipital and sphenoid bones and, as they grow, become firmly joined to the choanal region, and to any bone with which the growing tumor comes in contact, in addition to that of the area in which the growth began.

The nasopharyngeal fibroma is always formidable because of the difficulty of controlling the hemorrhage after the initial steps of operation have started bleeding, but before sufficient excavation can be made to approach the region from which the hemorrhage comes.

Nasopharyngeal fibroma occurs about fourteen times as often in boys as in girls, the great majority of them when they are between thirteen and sixteen years of age. About 21 per cent have occurred at fifteen years of age. The average age has been older in those few occurring in females. As the ossification between the sphenoid and occipital bones is completed by twenty-five years of age, the change in circulation then probably accounts for the fact that these tumors sometimes shrink away after twenty-five.

DIAGNOSIS

As to diagnosis, there is constant and increasing nasal obstruction. A polyp has a typical color and is easily movable, while fibroma is more nearly the normal tissue color and very firm. It cannot be pushed about, and it usually has a sessile base; often with a history of recurrent and profuse nosebleed. Though it be small, it can usually be seen with a mirror in the nasopharynx; if large, it can easily be palpated. It usually projects into the nasopharynx far enough to be easily seen. It may bulge the soft palate forward, and may extend into the oropharynx, one case in the literature being mentioned which extended into the esophagus.

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